# Refine Search

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| Terms | Documents |
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| antioxidant same (liver adj3 function) same (alcohol or ethanol) | 8 |

**Database:** US Pre-Grant Publication Full-Text Database; US Patents Full-Text Database; US OCR Full-Text Database; EPO Abstracts Database; JPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

**Search:** L9

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## Search History

**DATE: Thursday, November 02, 2006** Purge Queries · Printable Copy · Create Case

| Set Name side by side | Query | Hit Count | Set Name result set |
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| *DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR* | | | |
| L9 | antioxidant same (liver adj3 function) same (alcohol or ethanol) | 8 | L9 |
| L8 | antioxidant same (liver adj1 damage) same (alcohol or ethanol) | 14 | L8 |
| L7 | antioxidant adj10 liver adj10 (alcohol or ethanol) | 4 | L7 |
| L6 | antioxidant adj5 liver adj5 (alcohol or ethanol) | 1 | L6 |
| L5 | antioxidant same liver same (alcohol or ethanol) | 286 | L5 |
| L4 | hydrangea same (alcohol or ethanol) | 72 | L4 |
| L3 | hydrangea same liver | 5 | L3 |
| L2 | hydrangea same liver same (alcohol or ethanol) | 0 | L2 |
| L1 | hydrangea | 973 | L1 |

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**Search Results** - Record(s) 1 through 5 of 5 returned.

☐ 1. Document ID: US 20060153794 A1

L3: Entry 1 of 5 File: PGPB Jul 13, 2006

PGPUB-DOCUMENT-NUMBER: 20060153794
PGPUB-FILING-TYPE:
DOCUMENT-IDENTIFIER: US 20060153794 A1

TITLE: Hair tonics and method of screening the same

PUBLICATION-DATE: July 13, 2006

INVENTOR-INFORMATION:

| NAME | CITY | STATE | COUNTRY |
|---|---|---|---|
| Hibino; Toshihiko | Yokohama-shi | | JP |
| Tsuji; Yumiko | Yokohama-shi | | JP |
| Soma; Tsutomu | Yokohama-shi | | JP |
| Denda; Sumiko | Yokohama-shi | | JP |
| Nakanishi; Jotaro | Yokohama-shi | | JP |
| Takahashi; Tadahito | Yokohama-shi | | JP |
| Umishio; Kenichi | Yokohama-shi | | JP |
| Kobayashi; Koji | Yokohama-shi | | JP |

US-CL-CURRENT: 424/74; 424/774

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw D

☐ 2. Document ID: US 20050106267 A1

L3: Entry 2 of 5 File: PGPB May 19, 2005

PGPUB-DOCUMENT-NUMBER: 20050106267
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20050106267 A1

TITLE: Zeolite molecular sieves for the removal of toxins

PUBLICATION-DATE: May 19, 2005

INVENTOR-INFORMATION:

| NAME | CITY | STATE | COUNTRY |
|---|---|---|---|
| Frykman, Gregory K. | Washington | DC | US |

Gruett, Glenn H. New London WI US

US-CL-CURRENT: 424/684

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw D

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☐ 3. Document ID: US 20040022758 A1

L3: Entry 3 of 5 File: PGPB Feb 5, 2004

PGPUB-DOCUMENT-NUMBER: 20040022758
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20040022758 A1

TITLE: Hair tonics and method of screening the same

PUBLICATION-DATE: February 5, 2004

INVENTOR-INFORMATION:

| NAME | CITY | STATE | COUNTRY |
|---|---|---|---|
| Hibino, Toshihiko | Yokohama-shi Kanagawa | | JP |
| Tsuji, Yumiko | Yokohama-shi Kanagawa | | JP |
| Soma, Tsutomu | Yokohama-shi Kanagawa | | JP |
| Denda, Sumiko | Yokohama-shi Kanagawa | | JP |
| Nakanishi, Jotaro | Yokohama-shi Kanagawa | | JP |
| Nakanishi, Jotaro | Yokohama-shi Kanagawa | | JP |
| Takahashi, Tadahito | Yokohama-shi Kanagawa | | JP |
| Umishio, Kenichi | Yokohama-shi Kanagawa | | JP |
| Kobayashi, Koji | Yokohama-shi Kanagawa | | JP |

US-CL-CURRENT: 424/74; 424/725, 424/729, 424/750, 424/757, 424/766, 424/769

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw D

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☐ 4. Document ID: US 20020110605 A1

L3: Entry 4 of 5 File: PGPB Aug 15, 2002

PGPUB-DOCUMENT-NUMBER: 20020110605
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020110605 A1

TITLE: Liver function protecting or improving agent

PUBLICATION-DATE: August 15, 2002

INVENTOR-INFORMATION:

| NAME | CITY | STATE | COUNTRY |
|---|---|---|---|
| Nakagiri, Ryusuke | Tsukuba-shi | | JP |

| | | |
|---|---|---|
| Kamiya, Toshikazu | Tsukuba-shi | JP |
| Hashizume, Erika | Tsukuba-shi | JP |
| Sakai, Yasushi | Inashiki-gun | JP |
| Kayahashi, Shun | Tsukuba-shi | JP |

US-CL-CURRENT: 424/725

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Drawing D...

☐ 5. Document ID: US 4339442 A

L3: Entry 5 of 5 File: USPT Jul 13, 1982

US-PAT-NO: 4339442
DOCUMENT-IDENTIFIER: US 4339442 A

TITLE: Gynosaponins, their use and a process for preparing the same

DATE-ISSUED: July 13, 1982

INVENTOR-INFORMATION:

| NAME | CITY | STATE | ZIP CODE | COUNTRY |
|---|---|---|---|---|
| Takemoto; Tsunematsu | Tokushima | | | JP |
| Arichi; Shigeru | Toyonaka, Osaka | | | JP |
| Arihara; Shigenobu | Tokushima | | | JP |
| Nakajima; Tadashi | Ibaraki | | | JP |
| Okuhira; Megumi | Mishima-gun, Osaka | | | JP |
| Uchida; Yoshihiro | Taisho-ku, Osaka | | | JP |

US-CL-CURRENT: 514/26; 435/52, 536/4.1, 536/5

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Drawing D...

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| Terms | Documents |
|---|---|
| hydrangea same liver | 5 |

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L4: Entry 33 of 72 File: JPAB Aug 22, 2000

PUB-NO: JP02000229873A
DOCUMENT-IDENTIFIER: JP 2000229873 A
TITLE: ALPHA-GLUCOSIDASE INHIBITOR

PUBN-DATE: August 22, 2000

INVENTOR-INFORMATION:
NAME COUNTRY
KAWABATA, JUN
KASAI, TAKANORI

ASSIGNEE-INFORMATION:
NAME COUNTRY
NIPPON SYNTHETIC CHEM IND CO LTD:THE

APPL-NO: JP11030720
APPL-DATE: February 9, 1999

INT-CL (IPC): A61K 35/78; A61P 3/10; A61P 43/00

ABSTRACT:

PROBLEM TO BE SOLVED: To obtain the subject inhibitor having strong α-glucosidase inhibitory activity, being readily taken by making the inhibitor include a plant belonging to the family Rosaceae as an active ingredient.

SOLUTION: This inhibitor contains a plant belonging to the family Rosaceae as an active ingredient. For example, wild strawberry, rose, Chrysosplenium grayanum, Hydrangea paniculata, Rubus phoenicolasius, etc., may be cited as the plant belonging to the family Rosaceae. Though the plant belonging to the family Rosaceae is used in an ordinary state, further dried and powdered or ground in water into a slurried state and the slurried plant may show glucosidase inhibitory activity, an extract obtained by using water and/or an alcohol preferably provides stronger α-glucosidase inhibitory activity. An extract obtained by using a mixed solution of water and an alcohol is especially preferable. Ethanol is preferable as the alcohol. The weight ratio of water/the alcohol is preferably 1/100 to 100/1, more preferably 1/50 to 50/1.



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L8: Entry 10 of 14 File: USPT Jan 30, 2001

DOCUMENT-IDENTIFIER: US 6180139 B1
TITLE: Composition and method for treating nonalcoholic steatohepatitis

Abstract Text (1):
Nonalcoholic steatohepatitis (NASH) is a disease of the liver characterized by inflammation and damage to the liver cells. Typically, steatohepatitis involves inflammation of the liver related to fat accumulation, and mimics alcoholic hepatitis but is observed in patients who seldom or never consume alcohol. Nonalcoholic steatohepatitis can lead to serious liver damage, and ultimately cirrhosis. The present invention provides methods and compositions useful for the treatment or alleviation of nonalcoholic steatohepatitis and the pharmaceutical formulations for their administration to a human. Specifically, compositions comprised of lecithin, antioxidants and vitamin B complex are administered parenterally, most preferably by oral administration. Specific therapeutic formulations include admixtures of these compounds and specific dosage formulations include daily oral administrations of these compounds in tablet or powder forms.

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L10: Entry 45 of 68 File: USPT Nov 9, 2004

DOCUMENT-IDENTIFIER: US 6814951 B1
TITLE: Acetaldehyde and malondialdehyde protein adducts as markers for alcohol liver disease

Brief Summary Text (7):
Several studies have suggested that chronic ethanol consumption induces hepatic lipid peroxidation which in turn, generates malondialdehyde. MDA is toxic, mutagenic, and inactivates enzymes due to modification of lysine residues. MDA protein adducts have been detected in the liver following administration of agents that promote lipid peroxidation such as carbon tetrachloride, iron overload, and more recently chronic ethanol feeding. It has been shown to form an adduct with a lysine residue (.epsilon.-amino group) of proteins and that MDA reacts with a primary amine to give a 1:1 Schiff base. For further information about MDA protein adducts see Houglum et al., J. Clin. Invest., 86: 1991 (1990) incorporated herein by reference.

Brief Summary Text (10):
It is yet another object of the invention to provide a marker for alcohol liver damage which can be used to indicate the presence of liver disease, or other diseases with increased lipid peroxidation, lipids and/or or acetaldehyde which can include but is not limited to atherosclerosis or fat content for animals.

Brief Summary Text (19):
In addition to the above-identified novel hybrid adducts, the adducts of the invention have several important immunological properties which can be exploited for further chemical and immunological assay procedures. Monoclonal and polyclonal antibodies have been produced which recognize these adducts and can be used to identify them as markers of alcohol liver disease or other diseases associated with increased lipid peroxidation, lipids, and/or acetaldehyde such as atherosclerosis and fat content for domestic animals.

Detailed Description Text (82):
Numerous studies in the literature have applied immunochemical techniques to indicate the presence of a variety of protein adducts in the livers of ethanol-treated animals. These would include acetaldehyde adducts, MDA adducts, and more recently hydroxyethyl radical-derived adducts. However, structural information and epitope characterization of these adducts are lacking, and quantitative data have not been reported. In contrast, the applicants have provided quantitative estimates for MAA adduct formation and proposed structures of the MAA adducts. Furthermore, the results indicate that MDA and acetaldehyde react together in a synergistic manner which demonstrates that MAA adduct formation would be favored over adducts formed with acetaldehyde of MDA alone and that MAA adducts may represent a major species of adducts formed in the liver during ethanol metabolism in vivo. Since both the covalent binding of acetaldehyde to proteins and increased lipid peroxidation have been proposed as possible mediators of ethanol-induced liver injury, MAA protein-adduct formation represents an event dependent on both mechanisms, suggesting a common or unifying process (i.e. MAA adduct formation) by which both mechanisms can contribute to alcohol hepatotoxicity.

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L10: Entry 55 of 68 File: USPT Aug 17, 1999

DOCUMENT-IDENTIFIER: US 5939535 A
TITLE: Acetaldehyde and malondialdehyde protein adducts

Brief Summary Text (7):
Several studies have suggested that chronic ethanol consumption induces hepatic lipid peroxidation which in turn, generates malondialdehyde. MDA is toxic, mutagenic, and inactivates enzymes due to modification of lysine residues. MDA protein adducts have been detected in the liver following administration of agents that promote lipid peroxidation such as carbon tetrachloride, iron overload, and more recently chronic ethanol feeding. It has been shown to form an adduct with a lysine residue (.epsilon.-amino group) of proteins and that MDA reacts with a primary amine to give a 1:1 Schiff base. For further information about MDA protein adducts see Houglum et al., J. Clin Invest., 86:1991 (1990) incorporated herein by reference.

Brief Summary Text (10):
It is yet another object of the invention to provide a marker for alcohol liver damage which can be used to indicate the presence of liver disease, or other diseases with increased lipid peroxidation, lipids and/or acetaldehyde which can include but is not limited to atherosclerosis or fat content for animals.

Brief Summary Text (19):
In addition to the above-identified novel hybrid adducts, the adducts of the invention have several important immunological properties which can be exploited for further chemical and immunological assay procedures. Monoclonal and polyclonal antibodies have been produced which recognize these adducts and can be used to identify them as markers of alcohol liver disease or other diseases associated with increased lipid peroxidation, lipids, and/or acetaldehyde such as athereosclerosis and fat content for domestic animals.

Detailed Description Text (2):
Alcohol liver disease (ALD) is a major problem in the United States. However, it is not understood why all alcoholics do not develop ALD. It is known that following alcohol catabolism in the liver, acetaldehyde (AA) is generated. At the same time, lipid peroxidation is increased resulting in the production of malondialdehyde (MDA). Both of which have been suggested to have a role in liver damage. This invention relates to the discovery of a novel adduct present in the liver which is a hybrid of malondialdehyde and acetaldehyde. According to the invention, these two products combine to form a highly immunogenic antigen adduct, denoted as malondialdehyde-acetaldehyde-adduct or (MAA). As used herein the term antigen shall encompass any foreign composition capable of illicitng an immune response and includes lipids, carbohydrates, peptides, proteins, or even ribo and deoxyribonucleic acids which contain an amino group. This novel protein adduct has been shown to be present in patients with alcohol liver disease and in rats chronically fed alcohol, and as such can serve as a marker for diagnosis, monitoring, and understanding the pathogenesis of liver disease.

Detailed Description Text (84):
Numerous studies in the literature have applied immunochemical techniques to indicate the presence of a variety of protein adducts in the livers of ethanol-

treated animals. These would include acetaldehyde adducts, MDA adducts, and more recently hydroxyethyl radical-derived adducts. However, structural information and epitope characterization of these adducts are lacking, and quantitative data have not been reported. In contrast, the applicants have provided quantitative estimates for MAA adduct formation and proposed structures of the MAA adducts. Furthermore, the results indicate that MDA and acetaldehyde react together in a synergistic manner which demonstrates that MAA adduct formation would be favored over adducts formed with acetaldehyde of MDA alone and that MAA adducts may represent a major species of adducts formed in the liver during ethanol metabolism in vivo. Since both the covalent binding of acetaldehyde to proteins and increased lipid peroxidation have been proposed as possible mediators of ethanol-induced liver injury, MAA protein-adduct formation represents an event dependent on both mechanisms, suggesting a common or unifying process (i.e. MAA adduct formation) by which both mechanisms can contribute to alcohol hepatotoxicity.

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L11: Entry 35 of 40 File: USPT Feb 6, 2001

DOCUMENT-IDENTIFIER: US 6184227 B1
TITLE: Salts of aminoimidazole carboxamide useful in the prevention and treatment of liver diseases

Brief Summary Text (3):
The methods involve administering to an individual consuming alcohol, therapeutic drugs and/or exposed to xenobiotic agents, an effective dose of a salt of aminoimidazole carboxamide with or without antioxidants, including, but not limited to vitamin E, vitamin C, vitamin A and its derivatives, glutathione, N-acetylcysteine or magnesium gluconate. In the practice of the invention, compositions containing salts of AICA are used to detoxify harmful and noxious agents or toxins, to inhibit bioactivation of agents to harmful electrophiles or free radicals, to inhibit suppression of cell-mediated or humoral immune mechanisms, to stimulate the regeneration of target cells of the damaged issue and to inhibit the failure of energy supply. Preferred compositions of the invention are those which specifically or preferentially inhibit tissue injury involving , but not limited to, hepatocytes, nonparenclymal cells, endothelial cells, pit cells and other cells lining the hepatic sinusoids and bile duets.

Brief Summary Text (30):
The present invention is also directed to a method for the prevention and treatment of liver damage caused by alcohol which involves administering a salt of AICA and an antioxidant selected from the group consisting of vitamin E, vitamin C, vitamin A and its derivatives, glutathione, N-acetylcysteine and magnesium gluconate, to an individual in need thereof.

Brief Summary Text (32):
The present invention is also directed to a method for the prevention and treatment of drug-induced liver disease which involves administering a salt of AICA and an antioxidant selected from the group consisting of vitamin E, vitamin C, vitamin A and its derivatives, glutathione, N-acetylcysteine and magnesium gluconate to an individual in need thereof.

Brief Summary Text (33):
The present invention further involves a method for the prevention and treatment of hepatotoxic effects of xenobiotics and/or toxins, which involves administering a salt of AICA and an antioxidant selected from the group consisting of vitamin E, vitamin C, glutathione, N-acetylcysteine and magnesium gluconate to an individual in need thereof.

Detailed Description Text (3):
It will be apparent to those skilled in the art that other salts of AICA or agents having antioxidant properties and inhibit liver injury and enhance liver cell regeneration, may be useful as therapeutic agents. Such additional compounds may be identified using liver cell injury assays described herein.

Detailed Description Text (4):
It may be that the ability of AICA salts alone or in combination with antioxidants, to detoxify harmful and noxious agents, to inhibit bioactivation of agents to harmful electrophiles or free radicals, to inhibit suppression of cell-mediated or

humoral immune mechanisms, to stimulate the regeneration of liver cells and/or to inhibit failure of energy supply, contribute to the efficacy or effectiveness for use in the prevention and treatment of liver injury caused by alcohol, therapeutically useful drugs as well as industrial and environmental toxins. These possible mechanisms of action are in no way meant to limit the scope of the invention and are presented purely for explanatory and/or illustrative purposes.

Detailed Description Text (9):
Acetate may be oxidized to carbon dioxide and water or converted by the citric acid cycle to other biochemically important compounds including fatty acids. NAD is a co-factor and hydrogen acceptor when alcohol is converted to aldehyde and further to acetate. The NADH generated shuttles into the mitochondria and changes the NADH:NAD ratio and redox state of the liver. The hydrogen generated replaces fatty acid as a fuel and is followed by triglyceride accumulation and fatty liver. The redox state of the liver changes, protein synthesis is inhibited and lipid peroxidation increases. Situnayake, R. D. et al., 1990, Gut 31:1311.

Detailed Description Text (13):
10-15% of alcohol is metabolized by a microsomal p450 ethanol oxidizing system (MEOS). P450-II-E1 is part of this system and is inducible by alcohol and by some drugs such a acetaminophen and xenobiotics. Lieber, C. S., 1995, N. Engl. J. Med. 333:1058. This accounts for the susceptibility of the alcoholic to drugs that are hepatotoxic and which, when given in therapeutic doses, can cause serious liver injury. Induction of P450-II-E1 increases oxygen consumption, acetaldehyde production and promotes lipid peroxidation. During microsomal peroxidation, injurious reactive oxygen radicals or free radicals are produced and initiate lipid peroxidation. Metabolism of ethanol requires an excess supply of oxygen, a requirement that is met through proliferation of endoplasmic reticulum and induction of MEOS. The activity of MEOS favors utilization of reduced nucleotides. An excess of reducing equivalents results in generation of reactive oxygen species.

Detailed Description Text (17):
Therefore, an intervention aimed at inhibiting the production of lipid peroxides and related free radical moieties, for example, TXA2, can be expected to exert a protective effect against alcohol-induced liver injury. Thromboxane A2 synthesis can be inhibited by imidazole compounds, for example, aminoimidazole carboxamide. Horrobin, D. F., et al., 1978, Med. Hypothesis 4:178-184; and Terano, S., et al., 1985, Adv. Prost. Thromb. Leuk Res. 15-315. In addition, AICA was found to have antioxidant activity and to increase superoxide dismutase activity. Muzces, G., et al., 1990, Acta Physiologica Hungaria 76:183-190.

Detailed Description Text (18):
In the present invention administration of salts of AICA results in inhibition of thromboxane A2, enhanced antioxidant defenses against lipid peroxides and free radicals and increased nucleotide synthesis. More specifically, administration of AICA orotate results in prevention and/or inhibition of alcohol induced-liver injury and regeneration of damaged liver tissue. AICA salts in combination with antioxidants in the present invention are also useful for the prevention and treatment of alcohol-induced liver injury.

Detailed Description Text (26):
The reactive metabolites produced by P450 enzymes, often electrophiles or free radicals, form covalent adducts with proteins, lipids and nucleic acids, leading to disruption of their function. Oxidative processes also appear to be of fundamental importance in the pathogenesis of cell damage. Lipid peroxidation is prominent in several types of drug induced hepatic injury. Electrophilic conjugates also complex with intracellular antioxidants such as glutathione and deplete their supply. Oxidant stress causes mitochondrial injury and leads to impairment of cellular energy production and adenosine triphosphate (ATP) depletion and eventually to

toxic liver injury. Mehendale H M et al., 1994, FASE J 8:1285.

Detailed Description Text (27):
As discussed supra, in Section 5.1, intervention with AICA salts alone or in combination with antioxidants results in increased blood flow by inhibiting TXA2-induced vasoconstriction, and inhibition of lipid peroxide and free radical production. More specifically, use of AICA orotate in the present invention results in prevention and/or inhibition of drug-induced liver toxicity, regeneration of damaged liver tissue, renewal of nucleotides and energy supply.

Detailed Description Text (37):
As discussed supra, in Sections 5.1 and 5.2, in the present invention, AICA salts alone or in combination with antioxidants are administered to prevent and inhibit the formation of noxious metabolites and free radicals. More specifically, use of AICA orotate in the present invention results in the prevention and inhibition of drug-induced liver toxicity, regeneration of liver cells and detoxifying of industrial environmental hepatotoxins.

Detailed Description Text (45):
Formulations suitable for parenteral administration include aqueous and non-aqueous isotonic sterile solutions, which contain buffers, antioxidants and preservatives. The formulations may be in unit dose or multi-dose sealed containers.

CLAIMS:

6. The method according to claim 1 further comprising antioxidant therapy selected from the group consisting of N-acetylcysteine, vitamin E, vitamin A and its derivatives, vitamin C, gluthathione, cysteine, methionine and 2-mercaptoethanol.

12. The method according to claim 7 further comprising antioxidant therapy selected from the group consisting of N-acetylcysteine, vitamin E, vitamin A and its derivatives, vitamin C, gluthathione, cysteine, methionine and 2-mercaptoethanol.

18. The method according to claim 13 further comprising antioxidant therapy selected from the group consisting of N-acetylcysteine, vitamin E, vitamin A and its derivatives, vitamin C, gluthathione, cysteine, methionine and 2-mercaptoethanol.

20. The method according to claim 19 further comprising antioxidant therapy selected comprising antioxidant therapy selected from the group consisting of N-acetylcysteine, vitamin E, vitamin A and its derivatives, vitamin C, gluthathione, cysteine, methionine and 2-mercaptoethanol.

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# Refine Search

**Search Results -**

| Terms | Documents |
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| L5 and hydrange$ | 0 |

**Database:** US Pre-Grant Publication Full-Text Database; US Patents Full-Text Database; US OCR Full-Text Database; EPO Abstracts Database; JPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

**Search:** L14

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## Search History

**DATE: Thursday, November 02, 2006** **Purge Queries** Printable Copy Create Case

| Set Name side by side | Query | Hit Count | Set Name result set |
|---|---|---|---|
| *DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR* | | | |
| L14 | L5 and hydrange$ | 0 | L14 |
| L13 | L5 and phyllodulcin | 0 | L13 |
| L12 | L10 and phyllodulcin | 0 | L12 |
| L11 | L10 and antioxidant | 40 | L11 |
| L10 | (lipid adj2 peroxidation) same liver same (alcohol or ethanol) | 68 | L10 |
| L9 | antioxidant same (liver adj3 function) same (alcohol or ethanol) | 8 | L9 |
| L8 | antioxidant same (liver adj1 damage) same (alcohol or ethanol) | 14 | L8 |
| L7 | antioxidant adj10 liver adj10 (alcohol or ethanol) | 4 | L7 |
| L6 | antioxidant adj5 liver adj5 (alcohol or ethanol) | 1 | L6 |
| L5 | antioxidant same liver same (alcohol or ethanol) | 286 | L5 |
| L4 | hydrangea same (alcohol or ethanol) | 72 | L4 |
| L3 | hydrangea same liver | 5 | L3 |
| L2 | hydrangea same liver same (alcohol or ethanol) | 0 | L2 |
| L1 | hydrangea | 973 | L1 |